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ELECTROMYOGRAPHIC STUDIES OF MUSCLE DYSFUNCTION IN INFECTIOUS POLYNEURITIS AND POLIOMYELITIS*

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BOSTON

THE similarity of clinical findings in acute infectious polyneuritis¶ and in poliomyelitis and the difficulties in differential diagnosis have recently been emphasized by several authors.[1, 2] The outstanding symptoms that are common to both diseases are motor weakness or paralysis and varying degrees of tenderness and pain in the muscles of the back and extremities. The distribution of the involvement is an important point in differentiation, for in poliomyelitis the typical weakness is segmental and asymmetrical in distribution, whereas in polyneuritis the extremities are usually symmetrically weakened, the most severe involvement occurring proximally. Minor sensory changes can usually be detected in cases of polyneuritis and the cerebrospinal-fluid findings with albuminocytologic dissociation are characteristic of this disease. The prognosis in poliomyelitis in the acute stage is uncertain, residual paralyses being frequent, whereas in polyneuritis residual palsies are rare. The progression of paralysis is also different in these diseases, for in poliomyelitis the weakness is maximal during the acute phase, whereas in polyneuritis there may be a gradual increase in the degree of involvement and a spread to new muscles late in the course of the disease. Further details of the clinical picture and pathologic lesions have been discussed in recent reviews.[3–8]

In view of the similarity of symptoms in these diseases, we have treated patients with polyneuritis with hot packs and have observed as effective relief of muscle pain and tenderness as we have in poliomyelitis. To elucidate further the similarities in the behavior of muscles, we have studied by electromyography the patterns of electrical activity in the affected muscles of a series of patients with polyneuritis. Comparisons have been made with similar recordings from muscles in poliomyelitis in an attempt to throw light on the problem of muscle spasm and other dysfunctions in these diseases. In some cases comparisons have also been made with the recordings from cases with traumatic lesions of peripheral nerve, since these give direct evidence of the electrical behavior of muscle during regenerative changes in nerve.

METHODS OF STUDY

Ten cases of infectious polyneuritis from the Neurological Service of the Massachusetts General Hospital have been studied during the acute and convalescent stages of the disease. These had the characteristic symptomatology already described, including albuminocytologic dissociation in the cerebrospinal fluid.

The affected muscles were examined for clinical evidence of tenderness or pain on stretching, and their functional strength was assessed by the usual manual tests. The electrical discharges from these muscles at rest and during active and passive motion were recorded by means of an ink-writing oscillograph. Surface electrodes were usually employed, but in some cases these were supplemented by coaxial needle electrodes inserted in the belly of the muscle. The details of the technic have been fully described.[9] Comparisons have been made with similar studies on 10 patients with poliomyelitis and on others with traumatic nerve lesions.

RESULTS

Resting Muscles

The first series of electromyograms were recorded from resting muscles in positions of complete relaxation. No electrical discharges are recordable from normal controls by this technic under such conditions.[10, 11] In patients with polyneuritis, although the muscles were tender to pressure and stretching, spontaneous discharges were found only in the pres-

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¶Also known as infectious neuronitis, infectious polyneuritis with facia diplegia, Guillain-Barré's syndrome and Landry's paralysis.

ence of motor weakness. In poliomyelitis, also, electrical activity at rest was correlated with muscular weakness and not with clinical signs of sensitivity, except in complete paralysis, when no discharges were observed. The electromyographic patterns were similar in these two diseases (Fig. 1). The discharges persisted in both conditions so long as improvement continued or until recovery was complete. This suggests that spontaneous electrical activity

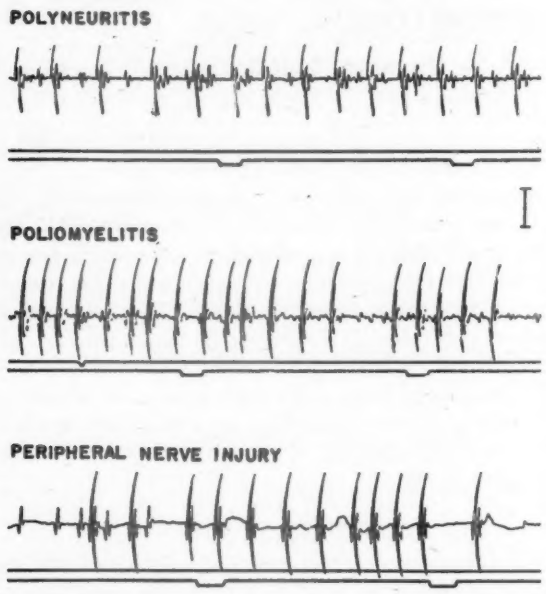

FIGURE 1. *Spontaneous Discharges from Partially Paralyzed Muscles at Rest as Recorded from Surface Electrodes by an Ink-Writing Oscillograph.*

The calibration is the same in all figures, the vertical line indicating 100 microvolts. The time interval is 1 second.

is a sign of a recovery process. Our studies on peripheral-nerve injuries corroborate this, for diphasic spikes of a similar appearance were found as an early indication of regeneration and are probably due to motor-unit activity, as suggested by Weddell, Feinstein and Pattle.[12] These spikes do not resemble the fibrillations of denervation, which are of much lower voltage and of too short a duration to be recordable by an ink-writing oscillograph.

A study of Figure 1 also reveals the striking similarity between the spontaneous electrical activity found in polyneuritis and poliomyelitis and the characteristic discharges present in muscles supplied by a regenerating motor nerve after traumatic lesion. This similarity is not surprising, since the electrical discharges from resting muscles in polyneuritis and in poliomyelitis are correlated with paresis and not with clinical spasm. They may well be associated with recovery of function in lower motor neurones.

In both polyneuritis and poliomyelitis, if the degree of paresis was unequal in a pair of opposing muscles, the greater electrical activity was found in the weaker muscle regardless of the degree of muscle tenderness (Fig. 2).

As a check on the type of recording obtained by surface electrodes in registering the spontaneous discharges of resting muscles, we have from time to time substituted coaxial needle electrodes. One example of such a comparison is given in Figure 3. It will be seen that the records are of essentially the same character.

Passive Stretching

The response of weakened muscles to quick, passive stretching consistently gave evidence of their hyperirritability in acute polyneuritis, as in poliomyelitis (Fig. 4). Instead of a few discrete spikes, as seen normally in controls, there were prolonged bursts of fairly high voltage discharges that persisted after the stimulus had ceased. These movements caused the patients some discomfort, and the muscle tension developed was similar to that seen in a pain reflex. Hyperirritability did not appear unless there was some appreciable weakness in the muscle tested, and when spontaneous discharges were present at rest these were increased by stretching. As clinical signs of muscle sensitivity lessened, the electrical activity in response to this stimulus also diminished.

Voluntary Contraction

The electromyograms of voluntary movements in polyneuritis revealed that protagonists and antagonists frequently contract simultaneously (Fig. 5), although this is not true of normal controls. This disruption of normal reciprocal innervation has also been demonstrated in poliomyelitis, and is of interest in relation to Kenny's[13, 14] concept of "inco-ordination." If the movements were painful, the electromyogram usually showed evidence of disordered reciprocal innervation. The inference from this observation is that the tension in the antagonistic muscles is a protective reflex serving to guard against excessive painful movement. Furthermore, this type of mass activation ceased with the decrease in muscle sensitivity.

In previous electromyographic analyses of this phenomenon in poliomyelitis, we[15] showed that in addition to simultaneous activation of opposing muscles there was synchrony between components of the discharge. This was also observed in cases of recovery from injuries to peripheral nerves, which suggested that the abnormality was that of the regenerative process. In the cases of polyneuritis, although simultaneous discharges were seen there were no clearly marked instances of synchrony of individual action potentials. In poliomyelitis this synchrony occurred only after severe paresis and only after complete paralysis in nerve injuries. In these cases dysfunction accompanying regeneration could be anticipated. In polyneuritis, however, the paresis was never so severe and the recovery was more

rapid; the absence of synchrony may therefore be explained by the lesser degree of neuronal damage.

Discussion

The similar distribution of muscle tenderness in polyneuritis and poliomyelitis is striking. Characteristically, in both diseases the muscles of the posterior neck, trunk and lower extremities are painful to stretching and deep pressure. It is also of interest that in both diseases hot fomentations are effective in relieving this symptom. In polyneuritis this might be explained by a sedative effect on hyperirritable sensory nerves, but in poliomyelitis no similar lesions are known. There are, however, definite pathologic changes occurring in the posterior-horn cells or nerve roots in both diseases that may be the basis of the similar response to heat. The pattern of electrical discharges obtained from affected muscles on passive stretching is also essentially the same in polyneuritis and in poliomyelitis, and is further evidence of a similar mechanism in both diseases.

Flexion contractures of the extremities are rarely seen as residual deformities in patients with polyneuritis, whereas these are not infrequent in poliomyelitis. Asymmetrical weakening of antagonistic muscles together with permanent paralyses may be responsible for these deformities in poliomyelitis. The possibility still remains that most of the residual limitation of joint movement is due to early immobilization with plaster and braces as commonly employed in poliomyelitis, whereas patients with polyneuritis rarely receive this orthopedic attention, being allowed to move freely in bed, and are usually given some form of heat for relief of sensitivity. It has been demonstrated by numerous observers[16–25] that without splinting and with intensive hot fomentations and therapeutic exercises in the acute stages, as recommended by Kenny, these deformities do not occur in poliomyelitis. The residual paralyses

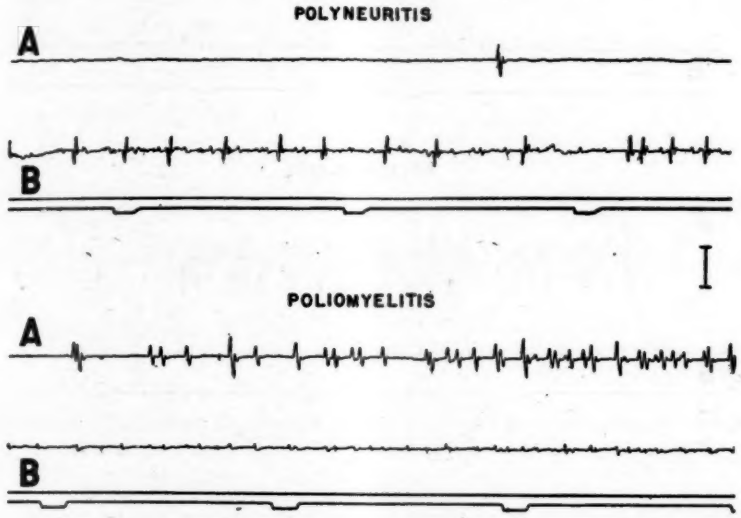

Figure 2. *Correlation of Spontaneous Discharges from Muscles at Rest with the Degree of Paralysis.*

The A *tracings are from the anterior tibialis muscle, and the* B *tracings from the gastrocnemius. The relative strengths were as follows: polyneuritis —* A, *90 per cent, and* B, *50 per cent; and poliomyelitis —* A, *30 per cent, and* B, *75 per cent.*

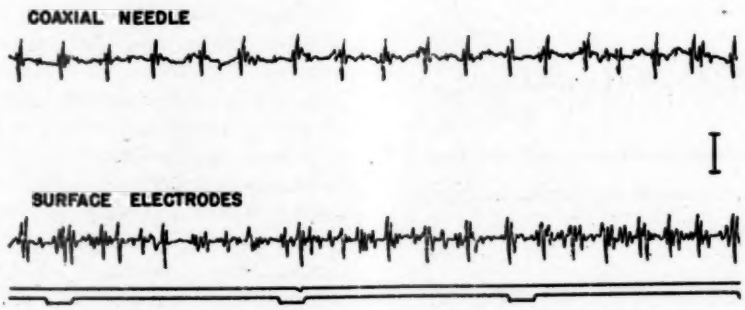

Figure 3. *Simultaneous Recording from Resting Muscle with Coaxial Needle and Surface Electrodes in a Case of Polyneuritis.*

from destruction of the anterior-horn cells cannot, of course, be influenced by any treatment applied to the muscles, although the function of remaining motor units can be brought to the maximum by physical therapy.

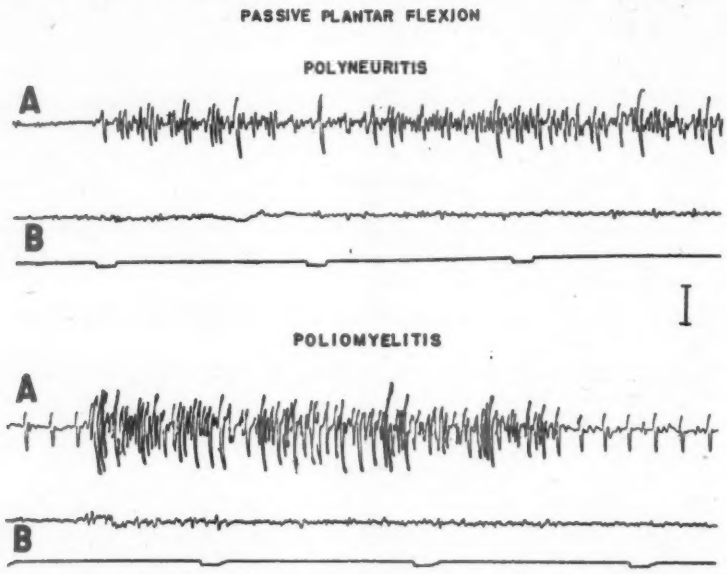

FIGURE 4. *Hyperirritability Elicited by Passively Stretching a Partially Paralyzed Muscle. The* A *tracings are from the anterior tibialis muscle, and the* B *tracings from the gastrocnemius.*

It has been suggested that spontaneous discharges from resting muscles in polyneuritis, poliomyelitis and traumatic nerve lesions are a sign of a regenerative process, since the electrical patterns are indistinguishable and these discharges are correlated with improving motor power. The histologic features of regenerating axons after traumatic nerve lesions are well known. The degenerative changes in peripheral nerves in polyneuritis have been described, and to account for recovery it is natural to assume that regeneration of axons takes place. In poliomyelitis no primary lesions in the peripheral nerves are generally recognized, although the virus may enter the cord by passage up an axon after experimental injection.[26] Axonal regeneration is, therefore, not thought to account for the improvement in motor strength that usually occurs. We have noted, however, that paretic muscles have diminished electrical excitability as measured by strength-duration curves, and that as power returns the excitability improves. This indicates that there are relatively severe although reversible lesions, presumably in the anterior-horn cells, and that secondary axonal changes may also occur. The electrical discharges probably represent returning function in motor units, but, of course, reveal no evidence of the types of histologic change that are taking place. The microscopical appearance of the axons is probably quite different in all three conditions under discussion, so that the term "regeneration" when used to describe our observations implies only a recovery process.

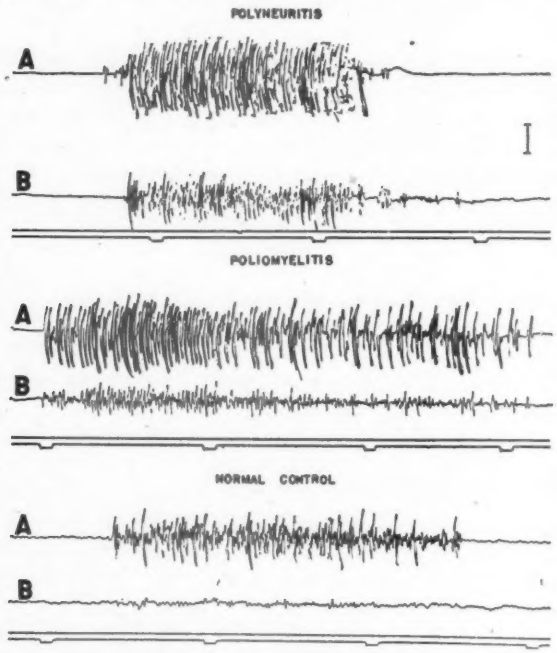

FIGURE 5. *Simultaneous Activation of Opposing Muscles on Voluntary Dorsiflexion.*

The A *tracings are from the anterior tibialis muscle, and the* B *tracings from the gastrocnemius. Simultaneous activation occurs in polyneuritis and poliomyelitis, but not in a normal control.*

The simultaneous activation of opposing muscles observed in polyneuritis and poliomyelitis might be thought of as a type of "inco-ordination," as described by Kenny.[13, 14] This is not, of course, inco-ordination in its usual meaning of abnormal muscular control in cerebellar disease. We prefer to interpret it as disruption of reciprocal innervation. In some

cases this disorder is apparently related to pain, the antagonistic muscle involuntarily contracting to prevent excessive movement. In poliomyelitis and in regenerating peripheral nerves after injury, we observed synchrony of action potentials, indicating severer disorganization of reciprocal innervation. In traumatic nerve lesions this probably represents abnormal axonal regeneration. There may also be similar abnormalities in the regenerative processes in poliomyelitis, although histologic evidence of this is lacking.

Summary and Conclusions

Electromyographic studies revealed the following similarities of muscle dysfunction in infectious polyneuritis and poliomyelitis: muscle tenderness and paresis are characteristic of both diseases, and the electrical abnormalities in both cases are correlated with weakness rather than with sensitivity; partially paralyzed muscles on stretching show hyperirritability, which can be relieved by hot fomentations; even at rest, paretic muscles are hyperirritable, discharging electrical potentials characteristic of regenerating motor units; and reciprocal innervation is frequently disrupted in polyneuritis and poliomyelitis, with resulting simultaneous activation of opposing muscles — this abnormality is probably secondary to painful motion.

The disorganization of reciprocal innervation is not so severe in polyneuritis as in poliomyelitis, for the simultaneous activation of opposing muscles in the former disease is not characterized by synchrony of component discharges.

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GERIATRICS: THE MEDICAL CARE OF THE ELDERLY*

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BOSTON

THE whole subject of geriatrics is relatively new. It can be compared to pediatrics, which was a blossoming subject when I was in medical school, but has now come to full fruition. The situation of the elderly has changed a great deal, because whereas a few years ago the average age of death was in the forties, a child born in the year 1943 may be expected to live to at least sixty-four years of age. The interesting thing is that the advances of medicine and public health have been directed almost entirely toward the early age group — that is, the age in which there has been improvement in mortality and morbidity statistics.

The man of forty to sixty-five lives no longer than he did fifty years ago, and there is a certain amount of evidence that he is no healthier. In other words, the present program of preventive medicine is enabling a great many people to live to sixty-five years but is not promising them anything when they do. It seems only fair that if they are to live as long as this, life ought to be made happier and healthier for them than it now is. Old age has been what one might call tolerated by the medical profession and by the public in general. No one is enthusiastic about old age. Certainly elderly people themselves are not particularly enthusiastic about it. In general, society has taken a negative and an entirely defeatist attitude toward old age. When the elderly are dead, society decrees a costly funeral and flowers.

As long as this negative, defeatist attitude continues, nothing is going to happen. Of course, part of this situation has been due to the fact that many of those who were looking for the alleviation of old age were not doing it from a medical or scientific point of view. All the folklore, all the tales about those who were interested in the elixir of youth — Ponce de Leon, in trying to find the Fountain of Youth, Ulysses, Sinbad the Sailor — show that these people were interested in only one thing, the re-creation of their sexual capacities, and not in the health of old age. This attitude has, of course, damaged the whole movement of geriatrics, when men are interested only in sex rejuvenation, and women only in keeping their charm, and, it will be noted, not usually their fertility.

I have no intention of discussing the damaging diseases of older people, — by which term is meant all those older than fifty-five and certainly those older than sixty, — such as advanced cancer, apoplexy and degenerative heart disease, about which at that stage nothing can be done. It may be said in passing that it is only by the study of the advanced stages of disease that one can gradually get back to a study of the origins of the disease; therefore, a certain amount of attention paid to cancer, even advanced cancer, apoplexy, degenerative heart disease or what not may bear fruit in the long-distant future. A few simple diseases, however, are particularly frequent in old age. Gallstones, for example, increase in frequency with advancing birthdays, and it is likely that if people lived to be one hundred and twenty they all would have gallstones. Certainly, autopsies performed on people of eighty or over show almost uniformly the presence of gallstones. This may have some effect on the present attitude toward the diet of older people, which is discussed below.

Another illustration of the diseases of old age is diverticulosis, which is increasingly frequent with advancing age. X-ray studies show that nearly all people over sixty have a few diverticulums. This condition may lead to an inflammation of one of the diverticulums — the clinical entity known as diverticulitis.

Inflammation of the intestines also seems to be on the increase in the elderly. Appendicitis is usually thought of as a disease of young people, and it is certainly extremely common in that age group, but it again becomes frequent after the age of sixty. Since gallstones occur in elderly people, it is often taken for granted that a stomachache is due to them or to diverticulitis, but a passing plea must be made for the consideration of appendicitis, which often takes the gangrenous form in people over sixty.

As the aging process, like the growing process, has been studied, it has been found that to keep the machine running, just the same as to keep the machine growing, there must be food and nourishment. Probably one of the greatest mistakes, by and large, has been to let the aged select their own diet, the latter depending on the condition of their teeth, whether they could chew it or not, depending perhaps on their pocketbooks and on the convenience of getting the food. It is certain that whereas in the family circle great attention is paid to the food for growing children, little attention is paid to the food for elderly people, even though they need it to continue their somewhat waning energies. To repeat, probably the most important thing in elderly people is the administration of the proper food in adequate quantity and quality. Here may be seen par excellence the necessity of giving vitamins.

*Presented at the annual meeting of the New Hampshire Medical Society, Manchester, May 11, 1943.

For many years, I was rather an off horse concerning many of the vitamins, and it has only been in the last few years, when vitamin concentrates have become available, that I have been able to convince myself of their value, particularly in people with a restricted diet, and particularly with the aged. On the subject of vitamins, the elements of the vitamin B complex are particularly indicated in older people, for two reasons. One is that the vitamin B complex — this refers to most of the factors in that complex — is easily used up in the human economy, in marked contrast to vitamins A, C and D, and has to be replaced frequently, one might say continuously. Furthermore, as a person gets older his carbohydrate intake is apt to be increased, and he needs certain components of the complex for catalysts of the carbohydrates to assist digestion.

In older people — perhaps because they are subject to two conditions, one of them gallstones and the other diverticulosis — it is the common experience that the administration of a high amount of fat and of roughage is often contraindicated. The difficulty with fats may have some relation to the existing gallstones, or it may have a simple relation to the fact that fats are much more difficult to digest than are any other food elements.

In connection with diverticulosis, it is usually evident that elderly persons do not take the high-roughage diet, which is so fashionable nowadays and which middle-aged women take with such enthusiasm. Such a diet often disturbs the digestion as people get to sixty and beyond, and it is usually advisable to restrict, at least moderately, the amount of roughage that goes into the diet.

I used to be one of those who objected strenuously to many of the digestive remedies that at one stage were so popular. As one studies elderly people, one appreciates that one of the things that does occur in them is the diminution in function or secretion throughout the body, and the same is true of the digestive tract. As one practices clinical medicine more and more, one changes one's ideas a little, and with the elderly I have entirely changed my ideas about the efficacy of some so-called "digestive assistants," foremost of which perhaps is the addition of hydrochloric acid to the diet. It is well recognized that as a person gets older he is apt to have less and less hydrochloric acid in his stomach and has more and more gas, and it can be shown clearly that if the reaction of the food and of the material in the stomach is changed, other changes are produced all the way down the intestinal tract. Hydrochloric acid is mentioned merely as one of the possible adjuvants to the therapy of older people, because it represents a principle of supplanting or supplementing the juices and functions that have been reduced.

At one time, I was scornful about the use of pepsin. I have found, however, that in the aged, as with hydrochloric acid, pepsin and even the pancreatic extract are often of real value. The same is true of the administration of bile, as bile salts or otherwise. These substances are not as a rule highly effective among younger people, but toward the age of sixty there is usually a deterioration in the secretions of the body or a reduction in their total amount.

It is certain that older people are much better off without the laxatives that have been given so widely in the past. They now seem to be reserved almost wholly for the elderly, the argument being, as always, "It doesn't make any particular difference." But it does make a difference, and the intestinal tract can be helped by the same procedures that are used in younger people and by the same attempts to do without laxatives. I am a great believer in mineral oil, but realize that since it absorbs vitamin A, the latter may have to be replaced. I am also a firm believer in colonic irrigation for elderly people, done about once a week.

Just as I have discussed the gastrointestinal system, one can take the other systems and go over them with the same point of view. There is one system that comes to the mind of everyone who deals with older people — namely, the endocrine glands.

Potassium iodide, which is so often given for arteriosclerosis in older people, is prescribed for them by most physicians simply because it seems to agree with them. As a matter of fact, iodine is an important part of the thyroid secretion, and the latter is stepped up by the addition of iodine to the thyroid nucleoprotein. Furthermore, many elderly people with deficient thyroid secretion are greatly invigorated by the judicial administration of thyroid.

The disturbances of the endocrine glands are not always the disturbances of diminution, because after sixty toxic goiter is seen, and sometimes it persists and creates a puzzle in diagnosis. It is not necessary, however, to do a metabolism test on every old man who has a rapid heart and is losing weight just because one such gentleman happened to have an exophthalmic goiter and was cured by a subtotal thyroidectomy.

By all odds, the most frequent disturbance in the thyroid gland is the diminution in the quantity or quality of the thyroid secretion. Glycosuria — not to be confused with true diabetes — is frequent in elderly people but often requires no treatment. Elderly men are naturally enthusiastic about the possibilities of testosterone. Some of them have been through the various operations for rejuvenation, including the substitution of goat glands, and have not been made particularly happy except, perhaps, psychologically at times. The modern endocrine hormones are decidedly potent, but they are to be used with a certain amount of discrimination, just as are the female hormones, which are so useful in the menopause and subsequently. These two sets of hormones are often helpful in the elderly, but not so much from the personal view of the man

who wants to be a Don Juan or the woman who wants to be a Venus as from that of the general condition and general health.

No thoughtful person who considers the subject of diseases of the prostate gland fails to realize that although much benefit has resulted from the surgical approach, the final solution will probably be the taking of a pill, either by mouth or by injection. When it is discovered how and why the prostate gland grows old, they will be given substances that will control the prostatic hypertrophy and render surgical treatment unnecessary. But this development is still in the realms of wishful thinking and of experiment.

The most frequent causes of death in the elderly are of course the degenerative diseases of the heart, blood vessels and kidneys, and this is the field in which one would like to make progress. It is true, however, that at the moment there is no method and no clue for lessening the mortality of these diseases.

Dr. Timothy Leary has made out an excellent case for the damaging effect of cholesterol in premature and devastating arteriosclerosis. He calls this effect atherosclerosis. There is a certain amount of evidence of one sort or another that supports this view. On the other hand, a good deal of the experimental work now being done is based on the influence of certain diets on rabbits and other animals. Rabbits are herbivorous creatures, whereas man will eat anything, including meat. Of course, when cholesterol, which is an animal fat, is added to the diet of a rabbit or indeed to other animals there may be some findings that are not easily transferred to man. There is no indication that Italians, who live on vegetable fats and oils in contrast to Anglo-Saxons, who live on the protein ones, grow old any more gracefully or have any fewer diseases than the rest of mankind.

As a matter of fact, it is too bad that the real answer cannot be found. It is fashionable to state that degeneration of the heart and blood vessels is due to the wear and tear of life. But if this matter is closely examined it will be found that certain people develop conditions or diseases the explanation of which is, "No, that isn't wear and tear; that is just rust."

Those who are interested in general hygiene tell us that it is a matter of this, that or the other thing. But when it comes right down to it, health seems to have a great deal to do with the care with which one picks one's ancestors. If this is done with discretion and they had normal cardiovascular systems, they are apt to transmit them to their descendants.

It is also necessary to assume as a part of the treatment of old age that there is something else to live for than negative security. It is perfectly true that the occupants of old men's or old women's homes live a long time, or at least live almost indefinitely, but they live rather as vegetables than as real human beings.

In order to pass one's old age gracefully, pleasantly and helpfully, one must have some sort of objective in life, and that objective should be a little bit more than outliving somebody else. As one observes older people, however, it is interesting to see that this is often the dominating factor in their lives. It may be a son-in-law who is particularly despised; or the attitude may be that of two brothers who checked off people as they died, hoping, distinguished as they were, to be the oldest twins ever graduated from Harvard College when they passed away.

I saw a gentleman of ninety one day when he was in high spirits. He was a physician and a member of the Old Vienna Club. He rubbed his hands gleefully and said, referring to a lifelong friend, a fellow member of the club, "He isn't going to last long."

I mention these things not to cause amusement, but because there is some value in the psychologic treatment of older people. They must have some incentive to live or they will, at times, literally curl up and die.

In 1914 a ninety-five-year-old lady called her family together and said: "I am an old woman, and I am going to die. I have taken to my bed." She also announced that she would give her brooch to so-and-so and her cameo to so-and-so. About that time World War I started and she became quite interested in reading the papers. The family rather neglected the nice old lady upstairs. Since they usually read the paper downstairs first, she would go downstairs and get the paper, and she became quite interested in how the war was going. She lived along all four years of the war, and then came the Treaty of Versailles. She then said that she did not think that the war had really proved anything. So she went to bed, summoned all her relatives and again said, "Now, I am going to die." But they said: "You can't put that over on us. You said that four years ago." But this time there was no World War I; there was nothing for this woman to live for, and she did what most older people do. It took her three weeks to run down, — it usually takes that long when old people take to their beds, — and that time she did die.

What I am pleading for is that older people must be treated psychologically as well as physically. I am no psychologist or psychiatrist, but when I went into the practice of medicine in Boston there was in practically every house on Beacon Street a room upstairs in which some old person lived and was never allowed to leave. People put lace caps on them or did something or other, and they vegetated there until they died. But one does not see that any more. The older people are not standing for it, and this is the first step in the treatment of geriatrics. These older people are being got out. Although I do not approve of their picking up gigolos and going dancing, at the same time a certain amount of that sort of thing is to be recommended. The family and the physician must have a certain amount of interest in older

people. They must have attention. Just today, one of your distinguished members came up here and talked in such an undertone that my old ears could not hear him, so I naturally switched off my attention. The younger people must talk loud enough so that we older ones can hear what they have to say.

I can do no better in closing than to tell a story concerning Dr. Stephen Smith, who was the first health officer of New York City. Because he was a health officer, he thought he knew all the principles of health and of righteous living. He was one hundred years old, and some people wanted to give him a dinner. I thought that was a terrible outrage. Then I thought, well, after all, he is a hundred years old, and he won't live long; he may have a good time, so why don't they do it? It may kill him, but what of it? They gave him the dinner.

Because of my curiosity about these things, I went over and talked to Dr. Smith, because I wanted to ask him about his ancestors. He had any number of ancestors on both sides of his family that had lived to be one hundred or one hundred and five. He didn't like my question very much. Then I saw his daughter, who had given up her whole life to looking out for this old man. She was a spinster of about seventy-five winters, and she was not particularly communicative. I said to her: "Now, when people live to be old, there is always some reason for it. They either don't smoke or do smoke; they either don't drink or do drink; and they either drink in the morning or in the evening, or something of that sort. What do you think about that?" But, she would not bite on that. Finally, I said to her, "What do you give your fine old father to eat?" There was a little smile that came over her face, and she said, "Well, I feed him mostly on milk, whiskey and flattery, and the last two seem to be the more important!"

264 Beacon Street

PURPURA ANNULARIS TELANGIECTODES*

Report of a Case

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PURPURA annularis telangiectodes, first described by Majocchi,[1, 2] in 1896, has appeared infrequently in the American literature. In 1915, MacKee[3] published a comprehensive review of the subject, along with extensive observations of a case of his own. In 1929, Scholtz[4] reported a case and reviewed 7 cases reported since MacKee's original review. In 1930 and 1936, Way[5,6] published case reports with histologic descriptions. In 1933, Levin and Tolmach[7] reported a case in which the main clinical pictures of the skin corresponded typically with the accepted pictures of purpura annularis telangiectodes, and in which an autopsy was performed. Unfortunately, the latter cannot be accepted unreservedly because no histologic study of the skin lesions was made, and the described clinical course and gross autopsy findings were those of bacterial endocarditis, in which event, purpuric lesions might be expected to occur. In 1942, Wise[8] published a review of the clinical and histopathological features of purpura annularis telangiectodes with its differential diagnosis from progressive pigmentary dermatosis (Schamberg[9]). The following case report seems justified because the clinical and etiologic investigation was more extensive than in any previously reported case.

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CASE REPORT

H. L. J., a 20-year-old unmarried woman, was first seen at the Hitchcock Clinic on March 26, 1942, complaining of skin lesions on the lower halves of the legs. For 5 or 6 months she had had oozing, pustular lesions on both ankles, and for 2 weeks she had noted maculopapular, circinate, symptomless lesions over the lower halves of the legs, accompanied by slight edema of the left ankle. On May 29, she was seen by a dermatological consultant, Dr. Arthur M. Greenwood, of Boston, who made the diagnosis of purpura annularis telangiectodes. She was next seen on July 9, at which time no essential change in the skin lesions was noted.

The parents and seven siblings were living and well, and the family history was not remarkable in any way. In her childhood the patient had had measles, mumps, whooping cough and chicken pox, without complications. There was no history of rheumatic fever or of operations, injuries, major illnesses or previous hospitalizations. She had graduated from high school 2 years previously and had been a telephone operator for 1 year. The menses had begun at the age of 12 and had always been regular.

Physical examination was not remarkable, except for the skin lesions. The cardiac impulse was detected 8 cm. to the left of the midline in the 5th intercostal space. The sounds and rhythm were normal. The height was 66 inches and the weight 143 pounds. The temperature was 98.6°F., the pulse 80, and the blood pressure 110/60. There were purpuric, circinate, ulcerating, scarring, pigmented lesions of the legs and ankles and slight edema of the left foot. Two blood Hinton and two blood Kahn tests were negative. The red-cell count was 4,350,000 and the hemoglobin 11.8 gm. (Klett method). The white-cell count was 5350, with 2 per cent eosinophils, 1 per cent stab neutrophils, 60 per cent segmented neutrophils, 32 per cent lymphocytes and 5 per cent monocytes. The hematocrit was 40 per cent. The sedimentation rate was 74 mm. in one hour, and at another time 55 mm. The bleeding time was 2 minutes and 40 seconds, and the clotting time was 3 minutes. The platelet count was 217,500. The prothrombin time was 32 seconds (normal, 30 seconds). The blood nonprotein nitrogen was 25 mg., the blood sugar 83 mg., the plasma chlorides 590 mg., the serum cholesterol

102 mg., the serum calcium 9.3 mg., and the serum phosphorus 3.3 mg. per 100 cc. Agglutination tests with Brucella organisms were negative. The plasma ascorbic acid was 1.5 mg. per 100 cc. A second-strength, intradermal tuberculin (P. P. D.) test was negative. A tourniquet test was negative. A radiograph of the veins of the left leg was performed after the injection of Diodrast into a superficial vein on the dorsum of the left foot. This showed normal visualization of the draining veins up to the femoral and external iliac veins. A radiograph of the chest was normal. An electrocardiogram was normal.

A biopsy of skin of the left leg was performed, the specimen being taken from an area showing telangiectasia, hemorrhage, pigmentation and atrophy. At the time of the biopsy, aerobic and anaerobic cultures were taken from the subcutaneous tissue, and these were sterile. Cultures on Sabouraud's medium were also sterile. Portions of the tissue were fixed in 10 per cent formalin and in Zenker's and Bouin's fluids, and the following stains were made: hematoxylin and eosin, Weigert's elastic tissue, Ziehl–Neelsen's, Masson's trichrome, and Turnbull-blue stain for iron. Serial sections were studied. A few capillaries showed hyaline thickening without other changes; others showed hyalinization of the walls accompanied by the formation of hyaline thrombi that completely occluded the lumens. There were scattered red cells in the connective tissue around such vessels, and an occasional widely dilated capillary with a ruptured wall was seen. In other areas there were proliferative intimal thickenings nearly occluding the lumens and, occasional round areas were seen that appeared to be recanalized, previously thrombosed vessels. These were surrounded by small collections of lymphocytes with occasional plasma cells and eosinophils. The capillaries around the hair follicles and sweat glands were particularly involved. Most of the other capillaries involved were in the superficial layers of the dermis. In some sections there was marked atrophy of the epidermis, which consisted of only two or three layers of cells; the immediate underlying dermis was fibrotic and showed hyalinized and thrombosed capillaries with extravasated red cells. Many of the sweat glands showed atrophic and fibrotic changes. In some of the hemorrhagic areas there were small collections of brown, granular pigment around the blood vessels, and these took the iron stain. The small arteries and veins of the subcutaneous tissue adjacent to the dermis were free of thrombotic and inflammatory changes. The elastic-tissue stain showed an apparent decrease in the elastic fibers of the dermis. No acid-fast bacilli were found in the sections stained by the Ziehl–Neelsen method. (The perinuclear vacuolization of the epidermis described by Way[8] appears to have no significance, since it has been seen in numerous sections of stratified squamous epithelium, including apparently normal tissues.)

Summary

A case of purpura annularis telangiectodes is reported.

Extensive laboratory tests gave essentially normal values with the following exceptions: the blood cholesterol was moderately low, and the sedimentation rate was markedly elevated.

Histologic study of the skin lesions revealed thromboses, inflammatory changes in and around small blood vessels, hemosiderin deposits and atrophy, all of which have been previously described.

This case furnishes no evidence that purpura annularis telangiectodes is a dermatologic manifestation of a cardiovascular disease.

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Addition. In the paper "Pathology, Clinical Manifestations and Treatment of Lesions of the Intervertebral Disks" by Dr. Albert Oppenheimer, which appeared in the January 27 issue of the *Journal*, the following sentence should have been added to the first footnote in column 1, "Awarded the first prize in the competition for the Pray and Burnham Awards offered by the Trustees of the New Hampshire Medical Society."

MEDICAL PROGRESS

BLOOD BANKS AND BLOOD TRANSFUSION (Concluded)*

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TRAUMATIC SHOCK, BURNS AND HYPOPROTEINEMIA

The importance of the plasma proteins in the treatment of shock, burns and hypoproteinemia has been reviewed recently by Janeway[64] in this series of articles, and will not be repeated here. It may be pointed out, however, that desiccated plasma and solutions of albumin were developed specifically for use by the armed services, and their greatest value lies in their stability at room temperature for prolonged periods of time and in their concentrated form, which allows shipment in a minimum amount of space. For civilian use it is more convenient and economical to preserve whole plasma in the frozen state, and such plasma is probably more suited for general use than are the desiccated or fractionated products. Liquid plasma stored at 4°C. or at room temperature maintains its effectiveness in restoring the osmotic pressure of the blood,[65] but loses several of its other properties and is therefore not so satisfactory as is the frozen product.

In the event that plasma is not available, transfusion of whole blood should not be withheld from burned or shocked patients merely because hemoconcentration exists. In such cases donor blood is relatively dilute in comparison with the recipient's blood, and transfusion tends to arrest the development of further hemoconcentration.[66]

Transfusion of whole blood is indicated in patients in shock when hemorrhage has been severe, and in burned patients when hemolytic processes[67] or inhibition of bone-marrow function has led to a lowering of the hematocrit.[68]

INFECTIONS AND INFECTIOUS DISEASE

Specific and nonspecific antibacterial and antitoxic substances may be transferred by transfusions of fresh blood or plasma and are frequently of considerable value in the treatment of infections and infectious diseases.[69] Attempts to increase the concentration of these immune substances in the transfused material have led to the use of convalescent serums, immunotransfusions and highly concentrated immune globulins prepared from convalescent human subjects or immunized animals. Convalescent serums and immune globulins have proved highly successful in the treatment of certain infections — for example, scarlet fever, measles, mumps and pneumococcal and influenzal infections.[65] Immunotransfusion has not been so generally successful, probably because of the fact that the concentration of antibodies developed in the donor as a result of vaccine injections are not so high as those already present in the recipient.[70] Lyons,[71] however, reported that transfusions of blood from properly selected donors were of considerable value in the treatment of hemolytic streptococcus infections.

The role of transfused leukocytes in combating infection has not been established. Donor leukocytes may aid in combating disease,[69] although it is not possible to elevate significantly the recipient's white-cell count by transfusion. Transfusion is of little or no value in the treatment of agranulocytic angina.[72] It is frequently indicated in acute and chronic infections to supply erythrocytes and plasma protein. Occasionally, the benefits derived from such supportive therapy are pronounced.

Although the immune globulin substances appear to be quite stable, complement, leukocytes and perhaps other important antibacterial substances rapidly degenerate in stored blood.[44, 55, 69] Consequently, fresh blood should be used for maximum effectiveness in the treatment of infections.

PRECAUTIONS

The importance of establishing absolutely the compatibility of blood before transfusion cannot be overemphasized. An error in blood grouping or crossmatching may — and all too frequently does — result in a severe transfusion reaction and death. Tests of compatibility should be performed only by adequately trained persons, and the materials and technics used must be scrupulously controlled. Excellent and concise descriptions of blood-grouping technics, with practical considerations of necessary precautions and sources of error, are to be found in the manual by Schiff and Boyd[73] on blood-grouping technic, which should be readily available in every laboratory performing such tests.

In contrast to the universally employed procedure of establishing donor-recipient compatibility in respect to the A, B, AB and O groups, tests for the Rh factor are not widely used, and until recently were performed only in a few research laboratories. The importance of the Rh factor as an isoantigen has recently been discussed by Hooker[74] and Wiener,[75] and it is apparent that most intragroup hemolytic transfusion reactions are caused by transfusion of Rh+ blood into sensitized Rh− recipients.[76] Even though the Rh factor is a poor antigen, and only 15 per cent of the white population is Rh−, the hazards associated with transfusion of Rh-incompatible blood

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are so great that tests for the Rh factor and transfusion of Rh-compatible blood should be made a routine procedure for all transfusions. Certainly, transfusions should never be given to pregnant women or to recipients receiving repeated transfusions unless Rh compatibility has been established.

Unfortunately, in vitro cross-matching tests — even with Landsteiner's test-tube technic and with incubation and chilling of the serum-cell mixtures — do not always detect potentially sensitive Rh– recipients. The avoidance of Rh-incompatible reactions can be ensured only by making tests for the Rh factor in the cells of both donor and recipient, and by transfusing Rh– recipients with Rh– blood. Such determinations require the use of potent Rh-agglutinating serum, which is still difficult to procure. Rh-factor determinations, however, can usually be obtained in centrally located laboratories.*

The indiscriminate use of so-called "universal donors" is dangerous, since transfusion of Group O blood may produce severe hemolytic reactions.[77] Hesse[78] reports that, in 46 cases, transfusion of universal-donor blood caused 20 deaths. He recommends that universal-donor blood be used only in cases of extreme emergency, and then only if the agglutinating titer of the donor serum for the recipient's cells is not higher than 1:32. Neutralization of isoagglutinins with specific Group A and Group B antigens has been recommended by Witebsky, Klendshoj and Swanson[79] and appears to make even high-titer Group O blood safe for transfusion into members of other blood groups. The pooling of several lots of plasma so dilutes and neutralizes the isoagglutinins contained in any one plasma that pooled plasma may be given without the hazard of producing intravascular agglutination.[80]

The rate at which blood may be transfused safely depends to a large extent on the functional integrity of the recipient's cardiovascular system. In normal persons as much as 650 cc. of blood has been given in three minutes without adverse reaction[81]; the rate of flow usually recommended is 40 to 60 drops per minute, or 250 cc. per hour.

Blood and plasma that have been refrigerated may be transfused safely without preliminary warming.[82] Indeed, mistaken attempts at warming may actually produce hemolysis or protein denaturation. Frozen plasma should be thawed at a temperature of 37°C., and the time used for thawing should not exceed thirty minutes. Slower thawing or thawing at room temperature results in the formation of a heavy fibrin precipitate.

Stored blood or plasma should be filtered before being transfused, since small aggregates of fibrin are prone to develop in even the best preservatives, and the introduction of such thrombi into the venous system may produce a fatal reaction.[83] Most filters tend to become clogged with these fibrin aggregates, with the result that they are frequently discarded and the blood or plasma is given without filtration. This is highly dangerous, since if there is enough precipitate to clog a filter, it will certainly clog the arterioles or capillaries somewhere in the recipient's body. Although not ideal, the glass-bead filter recommended by the British Medical Research Council[84, 85] has proved satisfactory at the Massachusetts Memorial Hospitals.

Every precaution must be taken to prevent the transmission of disease by transfusion. The mere performance of a serologic test for syphilis is not adequate, since highly infectious syphilis may exist before the reaction becomes positive and even before the primary lesion appears.[86–88] Eichenlaub and Stolar[89] state that in 41 cases of transfusion syphilis serologic examinations would not have aided in detecting the disease in 18 of the infected donors. Serologic testing of donors must be complemented by careful physical examination and questioning to exclude the possibility of latent or seronegative syphilis.

In view of the long latency of malaria, it may be unwise to use persons with a past history of this disease as blood donors, even though examination of a blood smear fails to reveal the presence of parasites.

The epidemic of jaundice following yellow-fever inoculations in the United States Army has been traced to the presence of small amounts of pooled human serum in the vaccine,[90] and it appears that the jaundice-producing agent is a filterable virus.[91] Similar attacks of jaundice have been produced by the transfusion of blood and of convalescent serum,[92, 93] and are presumably caused by a similar agent. The implications of these findings in the selection of blood donors are not yet definite, since donors of some of the jaundice-producing bloods are apparently healthy and there is no way of determining that their blood is icterogenic.

Transfusion Reactions

Hemolytic transfusion reactions are unfortunately much more frequent than is usually suspected, the average incidence being about 2 per 1000 transfusions, with a mortality of 1.4 deaths per 1000 transfusions.[94] These rates place the risk of blood transfusion in the same range as the risk of some major operations, a fact that is all too often neglected. Many physicians take elaborate precautions to safeguard against much less important hazards, but depend on a young intern or an inadequately trained technician to establish the compatibility of donor blood. Fortunately, transfusion of incompatible blood does not always result in serious complications,[95] but the usual course of events following such transfusions is intravascular agglutination and hemolysis, hemoglobinemia, hemoglobinuria, severe systemic symptoms, oliguria, anuria, uremia and death in at least half the cases.[78, 94, 96]

The pathogenesis of the renal failure that is the usual cause of death in these accidents is still ob-

*In the New England area, Rh-factor determinations and potent Rh agglutinating serum may be obtained from the Blood Grouping Laboratory (Children's Hospital, 300 Longwood Avenue, Boston).

scure. Considerable doubt has been cast on the theory that anuria and uremia are produced by mechanical blockage of the renal tubules by hemoglobin casts precipitated in acid urine, and on the belief that such blockage can be prevented by keeping the urine alkaline.[97, 98] Careful studies of kidneys from cases of fatal transfusion reactions have failed to account for the anuria on the basis of renal tubular obstruction. The primary lesion in such kidneys is a marked degenerative change in the convoluted and collecting tubules.[17, 99] Furthermore, large amounts of pure hemoglobin may be injected intravenously and excreted in acid urines without producing any decrease in renal function or any untoward reaction.[99, 100] Finally, maintenance of an alkaline urine in several typical hemolytic transfusion reactions did not prevent death.[99]

It appears probable that transfusion-reaction nephritis is produced by the action of some toxic or antigenic substance on the tubular epithelium rather than by mechanical blockage of the tubular lumens. The nature of this hypothetical toxin is suggested by the recent experiments of Bing,[100] who found that hemoglobin, methemoglobin and metmyoglobin could be excreted in either acid or alkaline urine without disturbance of renal function so long as the alkali reserve of the subject was normal. Production of acidosis by the intravenous injection of lactic acid or hydrochloric acid prior to the injection of methemoglobin produced typical transfusion-reaction nephritis, with anuria, uremia and death. Such findings suggest that fatal human reactions occur when methemoglobinuria develops in patients with markedly reduced alkali reserves, as in severe shock.

The extreme seriousness of hemolytic transfusion reactions and the fact that about half the patients developing anuria die make it absolutely essential that effective therapeutic measures be instituted immediately in any patient suspected of having such a reaction. In spite of the evidence that transfusion-reaction nephritis is not produced by mechanical blockage of renal tubules, abundant clinical and experimental evidence supports the practice of alkalinizing the urine in all suspected cases.[96, 101] To be effective, alkalinization must be accomplished rapidly, and this usually necessitates parenteral administration of alkalinizing agents. Because of the ready availability of sodium citrate in sterile ampoules in hospitals where transfusions are given, several authors have recommended the slow intravenous injection of 100 to 200 cc. of a 2.5 or 3.0 per cent solution of the drug, which, it is stated, produces a neutral or alkaline urine within fifteen minutes.[102, 103] More physiologic in action, safer and probably more effective in alkalinizing the urine are solutions of sodium lactate (⅙ molar) and sodium bicarbonate (4 to 6 per cent), which can be procured in sterile ampoules and which should be available for immediate use in any hospital where transfusions are administered. The oral administration of adequate amounts of sodium bicarbonate or other alkalies[102] produces an alkaline urine, but the length of time and the amounts required vary greatly in different persons. Numerous other therapeutic measures directed toward stimulating renal function and the secretion of urine have been advocated, but there is little evidence of their effectiveness.[104] Transfusion of fresh compatible blood as soon as possible after the hemolytic reaction has occurred does appear to be of some value, perhaps because of its effect in stimulating blood flow through the kidneys.[105]

It is evident from the confusion concerning the pathogenesis of transfusion nephritis and from the nonspecific nature and frequent failures of the numerous recommended therapeutic measures that the treatment of hemolytic transfusion reactions is unsatisfactory. It is better to avoid the necessity for such treatment by ensuring the compatibility of donor blood than it is to attempt treatment once a reaction has occurred.

Febrile reactions to transfusion are fairly frequent, and as a rule can be explained by the presence of pyrogens or impurities in solutions or by improperly cleaned glassware or tubing. Such febrile reactions may be serious in recipients who are critically ill or extremely anemic, and must be carefully guarded against by observing a scrupulous technic.

Allergic and anaphylactic reactions occur in 1 or 2 per cent of transfusions.[95] They vary in severity from mild urticaria to severe anaphylactic shock. Usually such reactions are mild and can be controlled by the injection of 5 to 10 minims of a 1:1000 solution of adrenalin. This type of reaction can be decreased in frequency by using only fasting donors, and it is now a routine requirement in many blood banks that donors must abstain from food for four to six hours before venesection is performed.

* * *

Blood banks and improved methods for the preservation of erythrocytes have made transfusion of whole blood a simple, economical procedure and have extended tremendously the therapeutic usefulness of transfusions. Blood plasma, originally a byproduct of the blood bank, now has its own particular fields of usefulness, and can be stored almost indefinitely without loss of therapeutic efficacy. The next great advance in transfusion therapy will no doubt be the preparation and use of purified fractions of the plasma proteins, — albumin (already in use), fibrinogen, thrombin, complement, specific immune globulins, isoagglutinins, enzymes and hormones,—each filling a particular therapeutic need.

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CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

Weekly Clinicopathological Exercises

FOUNDED BY RICHARD C. CABOT

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CASE 30071

PRESENTATION OF CASE

A forty-one-year-old housewife entered the hospital because of diarrhea.

The patient was in apparent good health until three and a half years before entry, when she developed numerous grossly bloody stools accompanied by crampy, low abdominal pain. Three years before admission she had an x-ray examination and was told that she had a "duodenal ulcer." On a Sippy diet and "injections" she had considerable relief for a period of two years. It is not known what the injections consisted of and whether there was an examination of the stools. Six months before admission she developed vague, distressing, abdominal discomfort just below the left costal margin. The pain was not severe and had no relation to meals. Four months before entry she had sudden, severe, prostrating, left subcostal pain and stayed in bed for seven weeks. Following this she was extremely weak; she was able to get up but stayed at home. She had intermittent attacks of subcostal, flank and lower costal pain on the left. About two weeks before admission she developed loose liquid stools alternating with normal stools. There was no blood, tarry material or recognizable food particles in the stools. On one occasion at that time she vomited foul-smelling material. She was taken to a community hospital where barium studies were performed. She had lost 50 pounds in four months. No additional information was available except that she had had a hysterectomy and appendectomy three years before entry.

Physical examination showed a well-developed, poorly nourished pale woman. There was a slight apical systolic murmur, but the heart and the lungs were not otherwise remarkable. The liver was palpable at the costal margin. In the left upper quadrant was a palpable, tender mass from which a tubular structure extended to the right upper quadrant. There was tenderness in the left subscapular region.

The blood pressure was 116 systolic, 68 diastolic. The temperature was 98.6°F., the pulse 75, and the respirations 20.

*On leave of absence.

Examination of the blood showed a white-cell count of 14,000, with 87 per cent neutrophils. The hemoglobin was 8.5 gm. per 100 cc. The blood protein was 4.4 gm. per 100 cc. The urine gave a + test for albumin. A gastric analysis showed free hydrochloric acid and was guaiac negative. The stools were guaiac positive. A blood Hinton test was negative.

A gastrointestinal series showed a rather large stomach. A wide fistulous tract, measuring 7 by 1.5 cm., led from the greater curvature of the stomach opposite the cardia to the splenic flexure of the colon (Fig. 1). Peristalsis of the stomach was active, and there was normal passage of barium

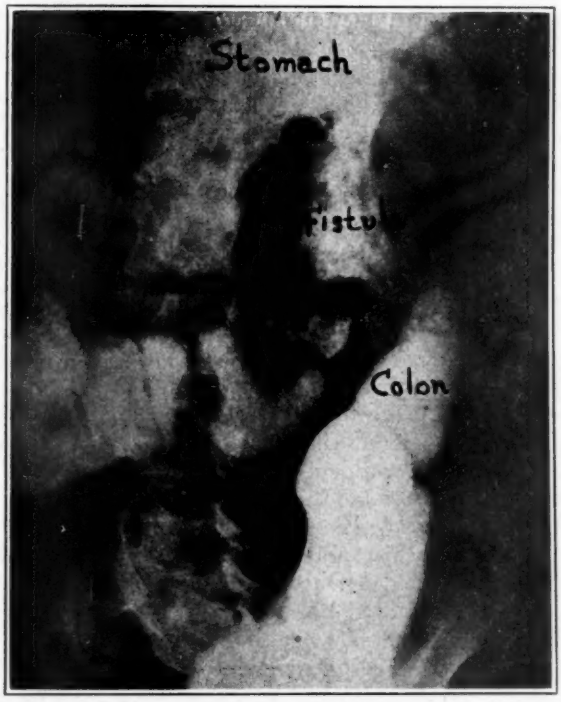

FIGURE 1. *Roentgenogram following a Barium Meal.*

through the pylorus filling a symmetrically nontender duodenal cap. Six-hour films showed a small amount of barium still in the stomach, mixed with food material. The barium was scattered throughout the small bowel, but little seemed to have passed through the fistulous tract to the left colon. The colon was outlined with barium from rectum to cecum. The barium was mixed with fecal material, apparently following barium examination of the colon before admission.

On the tenth hospital day an exploratory laparotomy was performed.

DIFFERENTIAL DIAGNOSIS

DR. ROBERT R. LINTON: Before reading the x-ray report it is well to pause and see what one can make out of this history. First of all, it seems to me that

we are dealing with a woman who was in poor condition. For instance, she had a hemoglobin of 8.5 gm., which is half what it ought to be. She had a low serum protein, 4.4 gm. per 100 cc., whereas it ought to be in the neighborhood of 6 gm. She had a + test for albumin in the urine. These findings, I think, indicate that the woman was seriously ill, and had a condition that had probably been going on for some time. The first indication of anything wrong was three and a half years before she was admitted, and at that time she apparently passed grossly bloody stools accompanied by crampy, lower abdominal pain.

The passage of blood in the stools may be due to several things. First of all it may be due to hemorrhoids. If it was due to hemorrhoids, one would not expect it to be associated with lower abdominal pain. One should never stop at that diagnosis without investigating the colon further, both by sigmoidoscopy and barium enema, which was not done. Occasionally one sees grossly bloody stools from a duodenal ulcer. She did have a gastrointestinal series at that time, and a diagnosis of a duodenal ulcer was made. Whether she had that or not, I am not certain, but I am sure that that is not what she was suffering from when she came into the hospital.

The next thing of interest in the history is that six months before admission she developed vague, distressing, abdominal discomfort below the left costal margin. We have for the first time some evidence of the localization of the lesion. Four months before entry she had sudden, severe, prostrating, costal pain on the left, and later there was a mass noted in the left costal region. What conditions can give symptoms in this region with a presumable mass? The first one is pancreatitis, in view of the fact that some extension of the mass toward the right upper quadrant was noted, which is the general distribution of the pancreas. It seems to me that a patient with severe left subcostal pain could very well have a pancreatic lesion, not a hemorrhagic pancreatitis but some form of pancreatic cyst or low-grade pancreatitis. Again she may have had a lesion in the stomach, as the stomach lies in the left upper quadrant. It may have been a carcinoma of the stomach, with a subacute perforation into the left upper quadrant that had sealed off. Another condition is a lesion in the splenic flexure of the colon, such as diverticulitis or carcinoma. One cannot forget also that the kidney and the spleen are in the left upper quadrant, but we have no evidence that either of these organs was involved. Furthermore, in the laboratory studies it is important to notice that the patient showed free hydrochloric acid in the gastric analysis. There was no blood in the gastric analysis, but the stool did give a positive guaiac test. From that I conclude that she was not suffering from carcinoma of the stomach and that the blood in the stools was more likely coming from a lesion in the colon rather than from one in the stomach.

I am a little confused about the x-ray report. Perhaps it is correct as written, but I should like to ask Dr. Robbins whether the barium went from the stomach into the splenic flexure. Apparently it did not go from the splenic flexure into the stomach.

DR. ROBBINS: This is the point at which the barium, as I understand it, was said to go through from the stomach to the splenic flexure. I think there is definite evidence of rigidity of the wall of the stomach. This is the fistulous tract, and in this projection it shows best on the spot films. It is distorted and fairly long. There is a narrowed area in the distal transverse colon at the point where the fistula enters.

DR. LINTON: It is obvious from the x-ray films that she had a gastrocolic fistula, and I think that one can pick out several confirmatory items in the history. For instance, it is stated that two weeks before entry she developed liquid stools alternating with normal stools. That does not necessarily mean that she had a gastrocolic fistula but suggests it. On one occasion there was vomiting with foul-smelling material. These two things ought to make one think of gastrocolic fistula in a patient with a lesion in the upper abdomen. It is perfectly obvious from these x-ray films, which help us in diagnosing the lesion, that the condition was not one of pancreatic or duodenal origin.

I believe that there are three possibilities to be considered: carcinoma of the stomach with perforation and a fistulous tract into the colon; carcinoma of the colon with the reverse taking place; and a benign lesion, such as a diverticulum, that had formed an abscess and had perforated into the stomach. I think the last mentioned is the least likely in view of the fact that the patient showed evidence of chronic disease—the low hemoglobin and low serum protein. It is up to me to decide whether she primarily had carcinoma of the colon or of the stomach, which is a fairly hard decision to make. The fact that there was rigidity in the stomach wall makes me favor a primary lesion in the stomach rather than one in the colon. Furthermore, a lesion that had been going on as many years as this one had, if it involved the colon, would ordinarily have given obstructive signs, which apparently were not present. This lesion was in a silent area in the stomach and could have gone undiagnosed for some time. When she had the first gastrointestinal series, the lesion was probably overlooked, since it was in the fundus.

My final diagnosis is carcinoma of the stomach, with perforation and the formation of a fistulous tract between it and the colon.

DR. ROBBINS: The rigidity in the wall of the stomach could just as well have been produced by extension from a lesion elsewhere or by an inflammatory process. There is a definite narrow area of ulceration in the colon from which the fistula extends.

DR. LINTON: Actually it makes little difference whether the lesion was in the stomach or in the colon, because both organs would have to be resected to relieve the patient. It is merely taking a long chance to see if one can guess the right one.

A PHYSICIAN: How do you explain the absence of occult blood in the stool?

DR. LINTON: I must admit that that is more indicative of a lesion in the colon than of one in the stomach. The free hydrochloric acid is also in favor of a colonic lesion rather than one in the stomach, but free acid does occur in carcinoma of the stomach, so one cannot draw any absolute conclusion.

DR. LELAND S. MCKITTRICK: Our reasoning was much like that of Dr. Linton. We were, however, more impressed by the radiologist's report than Dr. Linton was. We thought that the primary lesion was probably in the colon and that it had gone through into the stomach. We did not, however, feel strongly one way or the other.

We did a transverse colostomy and found an adherent mass in the left upper quadrant, which we did not thoroughly explore. We could not tell much about it but obtained the impression that it might be removable. About three weeks later she was reoperated on. During the interval she had seven or eight transfusions, but the hemoglobin at the end of that period was about 8.5 gm., practically what it was when she came into the hospital. At the second operation it was possible to take out the mass. We removed the greater part of the stomach, the spleen, about half the pancreas and the splenic flexure. Except for a thrombophlebitis involving the deep veins of the left leg, she made an uneventful convalescence.

CLINICAL DIAGNOSIS

Gastrocolic fistula.

DR. LINTON'S DIAGNOSIS

Carcinoma of stomach, with gastrocolic fistula.

ANATOMICAL DIAGNOSES

Adenocarcinoma of transverse colon, with extension into stomach.
Gastrocolic fistula.

PATHOLOGICAL DISCUSSION

DR. RONALD C. SNIFFEN: As Dr. McKittrick has indicated, the resection included the greater portion of the stomach, the transverse colon, the spleen and a segment of the pancreas. The transverse colon and the stomach were intimately bound together. When the former was opened, an annular tumor about 4 cm. in length was found that had replaced the mucosa. Superiorly, in the center of the tumor, a fistula about 2 cm. in diameter led into the stomach on the greater curvature. From the gastric aspect the fistula was found to be situated in the center of a mound produced by tumor infiltration of the stomach wall. The mucosa over this mound was ironed out but not destroyed, except at the portal of the fistula. To determine the origin of this tumor one must rely, in part, on the interpretation of the gross findings, and we believe that it arose in the transverse colon. Miscroscopically it proved to be a fairly well differentiated adenocarcinoma. The spleen was not involved, nor was the pancreas, although both organs were adherent to the mass.

CASE 30072

PRESENTATION OF CASE

A sixty-three-year-old woman was admitted to the hospital because of pain in the left leg.

The patient was in the hospital two years prior to admission because of a pathologic fracture through a benign giant-cell tumor of the left femur. The tumor was excised, leaving only the anterior medial and posterior walls of the cortex, which contained some of the original tumor. Grafts from the ilium were inserted. The patient was asymptomatic and moved around freely until two days before entry when she tripped over a rug and strained her leg, which necessitated the use of a cane. One month before admission she noted a mass in the left lower quadrant of the abdomen. There was no pain except for a "kink" on one occasion. She had no other symptoms.

She had been constipated for many years and had noted no change recently. Seven months prior to admission she noted a small amount of blood in the stools, but none since. A barium enema at that time was negative.

Physical examination showed a grapefruit-sized movable nontender mass in the left abdomen. It could not be moved into the pelvis or the flanks. There was anterior bowing and marked thickening of the left femur.

The blood pressure was 155 systolic, 75 diastolic. The temperature, pulse, and respirations were normal.

X-ray examination of the left femur showed progressive expansion in the involved area since the last examination. This was more marked in the lower portion, where numerous cyst-like cavities were seen that were divided by coarse trabeculae of bone. The bone grafts previously described were still in position and maintained their density. There was evidence of a fairly recent fracture of the anterior cortex at the upper extremity of the involved portion of the femur with anterior bowing, which was greater in degree than it was two years previously. There was no apparent soft-tissue mass in the left pelvis. A barium enema passed without delay from the rectum to the cecum. The proximal sigmoid was displaced laterally by a palpable mass

in the left side of the pelvis. The mass was extrinsic to the sigmoid and not grossly adherent to it. The rest of the colon was negative. A post-evacuation film showed contraction of the entire colon, with normal mucosal relief.

On the fifth hospital day an operation was performed.

Differential Diagnosis

Dr. Edward B. Benedict: The first question that occurs to me is a rhetorical one. I might ask why an endoscopist was asked to discuss a bone tumor. The first answer is a flattering one — possibly they thought that I am a good diagnostician. The second is that they wanted to "take me for a ride"; the third, that they could not get anyone else.

Dr. Ronald C. Sniffen: Let us assume that the first is correct.

Dr. Benedict: Could the pathologist be mistaken about the pathological diagnosis of benign tumor? You do not have to answer that now, Dr. Sniffen, but you might think about it.

Perhaps we might first look at the x-ray films.

Dr. Laurence L. Robbins: This is the bone lesion as it was originally seen. These films were taken in 1941. The location of the lesion is of interest. Apparently it is a trabeculated lesion with large cystic areas and appears to involve the entire bone, both the cortical and medullary portions. So far as I can see there is no evidence of break in the cortex other than what perhaps can be explained by a small fracture. I see no evidence of a soft-tissue mass. On this film, approximately three years postoperatively, you can see the changes that have taken place. The lesion has continued to expand, with loss of cortex in this area, but that does not necessarily mean that the lesion has become malignant. One wonders about it, however. There is soft-tissue swelling beyond a break in the cortex and evidence of fracture in the upper portion of the lesion, which probably explains the acute episode.

So far as the examination of the colon is concerned, the fluoroscopist definitely had the advantage, but I am inclined to think that the pressure is more on the transverse colon than on the sigmoid; that is, the transverse colon appears to be displaced in the distal portion. The sigmoid may have been displaced fluoroscopically.

Dr. Benedict: The record states that it was displaced laterally by a palpable mass in the left side of the pelvis.

Dr. Robbins: If there was a mass I should put it above the transverse colon.

Dr. Benedict: Was there a chest plate?

Dr. Robbins: Yes; it was negative.

Dr. Benedict: There are several things against a benign giant-cell tumor. Geschickter* states that no giant-cell tumor has recurred after primary radical resection. Furthermore, giant-cell tumors usually occur in the end of the bone. Fractures undoubtedly occur, but I wonder if one could expect two fractures in a recurrent giant-cell tumor. Such tumors occasionally become malignant, and I suppose that could have happened here.

What do you say, Dr. Sniffen, about the possibility of an erroneous microscopic diagnosis?

Dr. Sniffen: In this case I should say "No."

Dr. Benedict: Definitely?

Dr. Sniffen: Yes.

Dr. Benedict: By microscopic examination?

Dr. Sniffen: Yes.

Dr. Benedict: What do you think, Dr. Robbins? Is the bone tumor malignant by x-ray?

Dr. Robbins: The sections were taken at the time of the original films, and we do know that some giant-cell tumors become malignant. I do not know whether this is malignant now.

Dr. Benedict: I should like to relate the lesion to the abdominal mass, carrying out Dr. Richard Cabot's teaching of having one diagnosis to cover everything. In that case it would have to be a malignant tumor in the abdomen with metastases to bone. I thought of an ovarian tumor, but it was described as not being in the pelvis, and ovarian tumors seldom metastasize to bone. Furthermore, the plain film of the pelvis was said to have been negative. Tumors of the colon do go to bone, but not often. The barium enema was negative except for an extrinsic mass, which tends to rule out the colon.

The tumors that metastasize to bone commonly are those that originate in the prostate, breast, thyroid gland and kidney. I suppose that examination of the breast and thyroid gland was done and that a mass would have been noted. Tumors of the kidney frequently metastasize to bone, and the mass felt in the abdomen is consistent with a renal tumor. In addition, symptoms of the metastatic lesion rather than of the primary tumor may appear first. There is no record of x-ray films of the kidneys. The urine is not reported, and there was no pyelogram. Is there anything in the record about these?

Dr. Sniffen: The only finding recorded of remote significance is a rare red cell and rare white cell in the urine.

Dr. Robbins: I think that I can see the left kidney; it appears to be normal.

Dr. Benedict: Tumors of the lung and occasionally those of the stomach and esophagus go to bone, but we have no evidence of anything in the chest, stomach or esophagus. Lymphoma occasionally goes to bone, but usually to the vertebras, and this is not the x-ray picture of a lymphoma. Pathologic fractures are uncommon in lymphomas of the bone, and there should have been other evidences of lymphoma. From the x-ray standpoint this is apparently not an osteogenic sarcoma, because there is new-bone formation at the epiphyseal

*Geschickter, C. F., and Copeland, M. M. *Tumors of Bone.* 709 pp. Philadelphia: Lancaster Press, 1931. P. 326.

end. Furthermore, the patient is usually somewhat younger and pathologic fractures frequently occur.

I was going to put all my money on a hypernephroma with metastases to the femur, but Dr. Robbins and Dr. Sniffen believe that this is not a malignant bone tumor, and Dr. Robbins says that the left kidney outline is normal so far as he can tell. I do not know of any way to explain these two conditions on the basis of a benign giant-cell tumor except by making two diagnoses, and even then I cannot name a tumor of the left lower quadrant that is not in the pelvis, not in the flank and not in the colon. In spite of all the arguments against a malignant bone tumor I am going to say that this was a hypernephroma with metastasis to the femur.

Dr. Benedict's Diagnosis

Hypernephroma, with metastasis to femur.

Anatomical Diagnosis

Malignant lymphoma, lymphoblastic type, of stomach, with extension to transverse colon and involvement of regional lymph nodes.

Pathological Discussion

Dr. Sniffen: Dr. A. W. Allen operated on this patient, and I am sorry he is not here to discuss the case. I shall read a portion of his operative findings.

> The abdominal mass just below and to the left of the umbilicus was found to be a massive cancer in the region of the midtransverse colon. There were implants in the great omentum and in the gastrocolic ligament. Further exploration revealed that the source of the tumor lay in the stomach itself. There were nodes along the left gastric vessels, and nodes posteriorly to the celiac axis. The liver, however, was entirely free of disease; moreover there were no free peritoneal implants. After evaluation of the entire situation we decided that the tumor was resectable.

A total gastrectomy, a transverse colectomy and a splenectomy were performed.

I do not see how Dr. Benedict could possibly have made the diagnosis. I am afraid that he was the victim to illustrate an interesting gastric lesion. The two lesions were not related; the bone lesion was a benign giant-cell tumor.

The resected specimen was one of stomach, transverse colon and spleen. In the stomach there were two major masses, one on the greater curvature,

Figure 1. *Photograph of Stomach.*

Dr. Fletcher H. Colby: Would you expect a renal tumor to displace the colon the way this did?

Dr. Robbins: No; but I cannot be sure about the displacement of the colon. The fluoroscopist said one thing, but what I see on the films is transverse colon that may be displaced by a renal tumor. There is one peculiar thing — the gas shadow above the transverse colon appears to be in the mass displacing the colon; if one considers both films together, the gas shadow could be due to something in the stomach.

Clinical Diagnoses

Ovarian cyst.
Carcinoma of stomach.

about 10 cm. in diameter, and the other on the lesser curvature, about 7 cm. in diameter (Fig. 1). These were separated by several centimeters. In the rest of the mucosa there were several smaller nodules, and the rugae in general were large and moderately firm. Both masses invaded all coats of the stomach and had reached the serosa. The larger mass had invaded the transverse colon, which contained a small submucosal nodule. The mucosa over the tumors was atrophic, but apart from an area of central ulceration over the larger mass, it was intact. These lesions microscopically proved to be lymphoma of the lymphoblastic type.

It might be well to stress the fact that when one finds this picture — multiple tumors that originate in the stomach wall, protrude into the lumen and iron out the mucosa, with a central area of ulceration — one should seriously entertain the diagnosis of lymphoma. This also holds for the connective-tissue tumors, such as leiomyosarcoma and fibrosarcoma. Some of the regional lymph nodes were free from lymphomatous change, and some were involved. In an early lymphoma of the stomach one does not expect the lymph nodes to be involved, but in an advanced tumor such as this, it is more likely. The spleen was normal.

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REHABILITATION AND RE-EMPLOYMENT

The number of men rejected by the armed forces or honorably discharged with a "certificate of disability" is already great and will, of necessity, increase to an extent that may make it a difficult task for the civil community to absorb them adequately and gainfully. The proper rehabilitation and re-employment of these men is of the utmost importance if we are to avoid the tragic aftermath of the last war, and much of the responsibility for any future program rests on the civilian physician — for to him the discharged soldier will and should turn for help, guidance and assurance.

It is well to remember that the primary if not the sole objective of the induction stations of the armed forces is to procure a sufficient number of men who are physically fit for the rigors of general military service. The rejection of a selectee does not imply that he is incapable of living a normal existence or of being a useful civilian in his community, as he may well have been in the past. In a similar way, men are discharged from the armed forces with a "certificate of disability" because, for one reason or another, they are no longer able to stand the extreme stress and strain of modern warfare. Again there is no necessary implication that these men cannot return to a useful civilian life; they have been discharged solely because they are not fit for combat duty. It has not, however, been sufficiently stressed or publicized that many men are rejected or discharged for defects entirely compatible with normal civilian life. The man who has been rejected by an induction station or discharged from the armed forces may have a previously undiscovered disease that can and should be treated by his own physician. On the other hand, it must be emphasized that chronic diseases, either mental or physical, that have previously been known should gain no new significance or importance when the soldier returns to his home. The mere fact that he has been rejected or discharged must neither be a stigma against the man nor a cause of special anxiety to his family and friends, though all too frequently it is. The high-strung, tense person with frequent gastric complaints without organic basis presents no new problem when he returns home with the diagnosis "psychoneurosis, anxiety type." If he was an acceptable and useful member of society before, there is no reason why he should not continue to be such, provided his problem is understood and he himself is helped.

A man may have been gainfully employed for a period of years and yet be rejected by the armed forces for some psychiatric trait that makes him inefficient as a soldier; this fact renders him no less efficient as a civilian employee. Personality disorders that might well disqualify a man or cause his subsequent discharge again acquire no new significance. What may upset the family and may prejudice a future employer is that traits which had in civil life been regarded as erratic conduct or personal peculiarities were considered by the medical officers to be disqualifying for combat service. A man may be rejected for "healed tuberculosis," yet this diagnosis may thoughtlessly be translated into

"tuberculosis," with all its implications, and thereby the man's chances of future employment are materially lessened.

In short, the rehabilitation and especially the re-employment of the rejected selectee or the soldier or sailor discharged for medical reasons should be considered in the light of the fact that many men can function usefully in civilian life even though they cannot be effectively used in the armed forces. The chief role of the physician after determining that no previously unknown disease has been uncovered is to reassure the man, his family and his employer so that he may return to a useful function in civil life. This principle must likewise be appreciated by the man's friends and relatives and by the community at large. If it is not recognized that the physical and mental standards of the "fighter" are different from those of the civilian, we shall, once more, have the picture of the soldier or sailor who has fought for his country and returned only to be regarded as an outcast, a man who fought for a country existing only in his memory.

CUSHING'S *VESALIUS*

THE publication of Harvey Cushing's *A Bio-Bibliography of Andreas Vesalius* is an important landmark in the history of medicine. The book is the fruit of many years of study by a devoted disciple of the first modern anatomist, whose classical text of 1543, the *Fabrica*, is now acclaimed throughout a war-torn world, four hundred years after its publication.

Cushing's interest in Vesalius began as early as 1900, when he acquired Roth's biography, printed in 1892, and it continued up to a few weeks before his death in 1939. The writer recalls a summer day in 1939 spent with Cushing in New Haven. The dining room had been turned into a workroom, with the dining table covered with books. Cushing strove throughout the long, hot day, with only brief interruptions for meals on the rear piazza, and indeed far into the night, suffering awhile with much pain, but pushing the pace in a rush against time — time which only he could fully realize was growing so short. In spite of a desire for work, his innate kindliness would not cut short the leisurely honors to the departing guest the next morning; but both master and pupil seemed to sense that the parting might be the last and that the end of an eventful life could not be far distant. Fortunately for posterity, time was granted to Harvey Cushing to cast his book into reasonable completeness, and the final touches have been added by the loving and skillful hands of those nearest to him in his last year. The book may well be Cushing's most lasting contribution to medicine, living on in the heart of man long after his surgical treatises have passed into the forgotten world, vouchsafed to so many books. For Cushing not only wrote the life of Vesalius, but for many years lived the life of Vesalius. The impact of Vesalius on Cushing was a continuous one, and the trials, work and travels of the young genius of anatomical dissection cannot have been far from the conscious mind of Harvey Cushing as a daily experience. This is indeed reflected in his book, a faithful portrayal of all that is known of Vesalius — everything that he wrote and each place visited by a wandering spirit, who died by shipwreck, unknown and alone, at the age of fifty.

The form of this remarkable book is as unique as its contents. Cushing, the collector of the writings by Vesalius and of books that grew out of the works of the anatomist, saw the life of Vesalius written in the successive volumes from his pen. Using these, with their various printings and editions as a background, each volume being skillfully and completely described, Cushing wove the events of the man's career into the interstices, making a running whole. Thus both biographically and bibliographically the book is alive and readable, made more so by the numerous illustrations of title pages, frontispieces, anatomical figures, watermarks of papers and other details. To Dr. John F. Fulton, who expertly edits the book, one owes much for the bibliographical minutiae. Cushing's discerning eye holds the reader, his word-pictures telling the tale as only a master, thoroughly imbued with his subject, is privileged to do. Some of Cushing's finest writing occurs in this book, his farewell to a world made richer and finer by his presence and his published works. Well might someone write a bio-bibliography of Cushing himself, based on such a sound scheme for the depiction of man. In such a life the *Vesalius* may well be considered his crowning achievement.

MEDICAL EPONYM

Schmorl's Disease

Dr. Christian Georg Schmorl (1861–1932), discussed "Ueber Dehnungs- und Zerreissungsvorgänge an den Bandscheiben und ihre Folgen. [Events Preceding Expansion and Rupture in the Menisci and their Results]" at the twenty-second session of the Deutsche Pathologische Gesellschaft in Danzig, June 8 to 10, 1927. A report of his presentation may be found in the *Centralblatt für allgemeine Pathologie und pathologische Anatomie* (**40**:244-246, 1927). A portion of the translation follows:

> The normal turgor of the menisci may thereby so injure the spongiosa of the vertebral bodies as to lead to an abnormally marked indentation of the menisci. This is particularly the case if the spongiosa has deteriorated as the result of disease of the bone substance or the marrow, and its weight-bearing capacity damaged. . . .
>
> Under normal conditions also an injury to the spongiosa may result from the pressure due to turgor. This injury may be especially observed in young persons in whom endochondral growth is in process of conclusion or indeed has ceased. The injury is here limited to the region of the nucleus and is most probably to be attributed to a deterioration of the cartilage plates. . . .
>
> As a result of the indentation of the menisci there takes place an extension and thinning of the cartilage plates. Finally, excessive expansion, characterized by the formation of delicate fissures and rupture, occurs, usually in the nuclear region on both surfaces of one and the same meniscus at symmetrically located points. . . .
>
> . . . cavities of greater or lesser size arise in the spongiosa. . . .
>
> Cartilaginous nodules appear, which frequently lie at symmetrically situated points of the two surfaces of the menisci.

R. W. B.

MASSACHUSETTS MEDICAL SOCIETY

DEATHS

COUES — William P. Coues, M.D., of Brookline, died January 13. He was in his seventy-second year.

Dr. Coues received his degree from Harvard Medical School in 1894. He was appointed senior intern in surgery at the Massachusetts General Hospital in 1894. He was a member of the Massachusetts Medical Society and the American Medical Association.

CRONIN — Edward J. Cronin, M.D., of Brighton, died February 11. He was in his forty-seventh year.

Dr. Cronin received his degree from Tufts College Medical School in 1921. He interned at St. Elizabeth's Hospital and was staff secretary there at the time of his death. He was a member of the Massachusetts Medical Society and the American Medical Association.

His widow, five children, three sisters and five brothers survive.

EMERSON — Francis P. Emerson, M.D., formerly of Boston, died at the home of his daughter in Franklin on January 19. He was in his eighty-second year.

Dr. Emerson received his degree from Columbia University College of Physicians and Surgeons, New York, in 1886. He was formerly aural surgeon at the Massachusetts Eye and Ear Infirmary and the Massachusetts General Hospital. At one time he was instructor in otology at the Harvard Medical School. He was a member of the staffs of the Brooks Hospital and the Massachusetts Women's Hospital. He was a member of the Massachusetts Medical Society, the American Medical Association, the American Association for the Advancement of Science, the American Otological Society, the American Laryngological Society and the New England Laryngological and Otological Society.

One daughter survives.

KITE — Walter C. Kite, M.D., of Milton, died February 5. He was in his eightieth year.

Dr. Kite received his degree from the University of Pennsylvania School of Medicine, Philadelphia, in 1893. He served internships in hospitals in Philadelphia, Atlantic City and Wilkes-Barre, Pennsylvania. He was a member of the Massachusetts Medical Society and the American Medical Association.

His widow, a son, a daughter, a grandson and a brother survive.

RICHARDSON — Edward P. Richardson, M.D., of Brookline, died January 26. He was in his sixty-third year.

Dr. Richardson received his degree from Harvard Medical School in 1902 and for sixteen months before graduating was surgical house officer at the Massachusetts General Hospital. As a specialist in surgery he was at various times, assistant visiting surgeon to the Massachusetts General Hospital, and consulting surgeon to hospitals in Gardner, Milford, Brockton, Attleboro and Plymouth. In 1915, he served with the First Harvard Medical Unit at British General Hospital No. 22, near Etaples, France, as a temporary honorary major. In July, 1918, he was commissioned a captain in the United States Army Medical Corps. He was then assigned to Evacuation Hospital No. 30 and promoted to major in September and arrived in France with his unit on Armistice Day. In 1925, he accepted a full-time position at the Massachusetts General Hospital as chief of the West Surgical Service and associate professor of surgery at Harvard Medical School, later becoming John Homans Professor of Surgery. He held these positions until 1931, when he was obliged to withdraw from active practice because of ill health.

He was a member of the Massachusetts Medical Society, American Medical Association, American College of Surgeons, American Surgical Association, Southern Surgical Association, and Society of Clinical Surgery.

Three sons survive.

RILEY — Charles A. Riley, M.D., of Newton, died January 30. He was in his sixty-third year.

Dr. Riley received his degree from the Jefferson Medical College of Philadelphia in 1905. He was a member of the staff of the Brooks Hospital and a consultant for the public-health services in Boston and Newton. He was a member of the Massachusetts Medical Society and the American Medical Association.

His widow, a son and a daughter survive.

NEW HAMPSHIRE MEDICAL SOCIETY

DEATH

HYLAN — N. Wicker Hylan, M.D., a major serving as flight surgeon with the Air Corps of the Army of the United States in England, was killed on January 28 as the result of an aircraft accident. He was in his thirty-seventh year.

Dr. Hylan received his degree from Tufts College Medical School in 1935. He formerly practiced in Derry and went overseas in June, 1943. He was a member of the New Hampshire Medical Society.

His widow, four children, a sister and two brothers survive.

WAR ACTIVITIES

INDUSTRIAL MEDICINE

Precautions for the Handling of Chlorinated Compounds

The Halowax Products Division of the Union Carbide and Carbon Corporation has recently circularized to all its customers and to industrial-health agencies a pamphlet, "Precautions for Handling Chloronaphthalene, Chlorodiphenyl and Related Chlorinated Compounds." This action has been made necessary because of the frequency with which workers have developed acne after continuous exposure to these materials in the manufacture of synthetic waxes. The statement describes specific preventive measures, including engineering control procedures, selection of workers, health supervision of workers and personal-hygiene practices.

It is emphasized: "Experience has shown that, despite their toxic properties, these chlorinated materials may be safely used if proper control measures are observed. Under good operating conditions the appearance of acne should be reduced to a minimum, and easily brought under control. With these precautions, cases showing liver damage probably would never occur."

The company offers the services of its engineering department to advise on proper design for ventilating equipment needed to exhaust the fumes and vapors given off in both hot and cold processes of manufacture of these waxes. Advice will be given on all other protective measures that should be taken to avoid the hazards involved in handling the materials.

The pamphlet and additional information may be secured by writing the Halowax Products Division, 30 East 42nd Street, New York 17, New York.

MASSACHUSETTS DEPARTMENT OF PUBLIC HEALTH

COMMUNICABLE DISEASES IN MASSACHUSETTS FOR JANUARY, 1944

RÉSUMÉ

Diseases	January 1944	January 1943	Seven-Year Median
Anterior poliomyelitis	3	6	1
Chicken pox	2514	1362	1772
Diphtheria	26	5	17
Dog bite	479	451	579
Dysentery, bacillary	23	3	6
German measles	146	1165	67
Gonorrhea	387	289	357
Measles	1648	1886	1886
Meningitis, meningococcal	94	41	6
Meningitis, other forms	26	15	*
Meningitis, undetermined	14	5	*
Mumps	913	1113	771
Pneumonia, lobar	646	342	618
Salmonella infections	1	4	4
Scarlet fever	1207	1470	991
Syphilis	447	397	417
Tuberculosis, pulmonary	262	186	250
Tuberculosis, other forms	17	16	21
Typhoid fever	2	0	6
Undulant fever	6	0	2
Whooping cough	373	881	953

*Pfeiffer-bacillus meningitis only other form reportable previous to 1941.

COMMENT

Meningococcus meningitis is again markedly on the increase, the past month showing by far the largest number of cases of any January on our records. Lobar pneumonia was also at a high point, with an increase of 70 cases over the December and 422 over the November figure. Diphtheria, however, dropped considerably below December's rather high figure of 42.

GEOGRAPHICAL DISTRIBUTION OF CERTAIN DISEASES

Anterior poliomyelitis was reported from: Hudson, 1; Lenox, 1; West Springfield, 1; total, 3.

Diphtheria was reported from: Boston, 8; Dartmouth, 2; Fall River, 2; Ludlow, 1; Melrose, 2; New Bedford, 1; Quincy, 2; Somerville, 6; Taunton, 2; total, 26.

Dysentery, bacillary, was reported from: Boston, 3; Worcester, 20; total, 23.

Encephalitis, infectious, was reported from: Methuen, 1; Millbury, 1; total, 2.

Malaria was reported from: Bourne, 1; Camp Edwards, 3; Cushing General Hospital, 1; Fort Banks, 4; Fort Devens, 12; Fort Rodman, 2; Quincy, 1; total, 24.

Meningitis, meningococcal, was reported from: Ashfield, 1; Auburn, 1; Barnstable, 3; Boston, 16; Bourne, 1; Braintree, 1; Brookline, 2; Cambridge, 1; Camp Edwards, 2; Canton, 1; Chelsea, 2; Chelsea Naval Hospital, 1; Easthampton, 1; Edgartown, 1; Everett, 2; Fall River, 3; Fort Banks, 1; Fort Devens, 1; Hamilton, 1; Hanover, 1; Haverhill, 1; Hingham, 1; Holyoke, 3; Lawrence, 2; Lexington, 1; Lowell, 7; Malden, 1; Marlboro, 1; Medford, 1; Methuen, 1; Middleboro, 1; Milford, 1; Milton, 1; Newton, 1; North Adams, 1; Norwood, 3; Peabody, 1; Quincy, 3; Randolph, 1; Somerset, 1; Somerville, 3; Springfield, 2; Taunton, 1; U. S. Marine Hospital, 1; Wellesley, 1; Westfield, 2; Weston, 1; Worcester, 6; Wrentham, 1; total, 94.

Meningitis, Pfeiffer-bacillus, was reported from: Carlisle, 2; Framingham, 1; Lawrence, 1; Winchester, 1; total, 5.

Meningitis, pneumococcal, was reported from: Brookline, 1; Falmouth, 1; Leominster, 1; Lexington, 1; Malden, 1; Merrimac, 1; Salem, 2; Shrewsbury, 1; Somerville, 1; Southbridge, 2; Woburn, 1; total, 13.

Meningitis, streptococcal, was reported from: Methuen, 1; total, 1.

Meningitis, other forms, was reported from: Boston, 4; Brockton, 1; Easton, 1; Lynn, 1; total, 7.

Meningitis, undetermined, was reported from: Andover 1; Attleboro, 1; Boston, 7; Brockton, 1; Springfield, 2; West Springfield, 1; Worcester, 1; total, 14.

Salmonella infections were reported from: Brookline, 1; total, 1.

Septic sore throat was reported from: Amesbury, 3; Billerica 1; Boston, 6; Camp Edwards, 2; Easton, 1; Fall River, 1; Lawrence, 1; Newton, 1; Watertown, 1; Williamstown, 1; Winchester, 2; Winthrop, 1; total, 21.

Tetanus was reported from: North Reading, 1; total, 1.

Trachoma was reported from: Boston, 1; total, 1.

Trichinosis was reported from: Walpole, 1; total, 1.

Tularemia was reported from: Scituate, 1; total, 1.

Typhoid fever was reported from: Marblehead, 1; New Bedford, 1; total, 2.

Undulant fever was reported from: Boston, 1; East Bridgewater, 1; Northboro, 1; Norton, 2; Rutland, 1; total, 6.

CORRESPONDENCE

WAGNER–MURRAY–DINGELL BILL

To the Editor: The enclosed open letter has been sent to each of the New England congressmen, addressing each senator or representative by name. The letter expresses the point of view of the New England Dermatological Society with regard to Senate Bill 1161 and House Bill 2861, the so-called "Wagner–Murray–Dingell Bills."

FRANCIS M. THURMON, M.D., *Secretary*
New England Dermatological Society

520 Commonwealth Avenue
Boston 15

* * *

THE NEW ENGLAND DERMATOLOGICAL SOCIETY

January 25, 1944

Dear ————:

The New England Dermatological Society, with ninety-four specialist members from the six New England states, has studied Senate Bill 1161 and House Bill 2861, the so-called "Wagner–Murray–Dingell Bills," now before the Congress and respectfully submits its views on the proposed legislation.

At a regular meeting of this society held in Boston on December 8, 1943, the Society, by unanimous vote, heartily disapproved the content of S. 1161 and H. 2861 and without equivocation stated, "We do not believe these bills provide for the sound development of a national health program."

Agencies already exist to take care of the medical needs of all our people, and to improve their individual health. If only full advantage is taken of these agencies, creation of new agencies — such as projected by the Wagner–Murray–Dingell Bills — will but interfere with the orderly and diligent care of our sick. Let the present pattern of American Medicine continue to function. It has been honest. It will continue so and will adjust itself to the needs of our people.

As our representative in Congress we desire to point out to you our attitude on S. 1161 and H. 2861 and respectfully request that you give our point of view the serious consideration which it merits.

[Signed] Jacob H. Swartz
JACOB H. SWARTZ, M.D., *President*
[Signed] Francis M. Thurmon
FRANCIS M. THURMON, M.D., *Secretary*

520 Commonwealth Avenue
Boston 15

NOTICES

NEW ENGLAND HOSPITAL FOR WOMEN AND CHILDREN

The monthly clinical conference and meeting of the staff of the New England Hospital for Women and Children will be held on Thursday, March 2, in the classroom of the Nurses' Residence at 7:15 p.m. Dr. Harold A. Kent will speak on the subject "Why Is the Mouth Important?"

(*Continued on page xi*)

NOTICES (*Continued from page 208*)

SOCIETY MEETINGS AND CONFERENCES

CALENDAR OF BOSTON DISTRICT FOR THE WEEK BEGINNING THURSDAY, FEBRUARY 24

THURSDAY, FEBRUARY 24

*9:00–10 a.m. Medical clinic. Dr. S. J. Thannhauser. Joseph H. Pratt Diagnostic Hospital.

FRIDAY, FEBRUARY 25

*9:00–10:00 a.m. Visualization of the Biliary Tract. Dr. Charles G. Mixter. Joseph H. Pratt Diagnostic Hospital.

SATURDAY, FEBRUARY 26

*10:00–11:30 a.m. Medical staff rounds. Peter Bent Brigham Hospital.

MONDAY, FEBRUARY 28

*12:15–1:15 p.m. Clinicopathological conference. Peter Bent Brigham Hospital.

TUESDAY, FEBRUARY 29

*9:00–10:00 a.m. Medical clinic. Dr. S. J. Thannhauser. Joseph H. Pratt Diagnostic Hospital.

*12:15–1:15 p.m. Clinicoroentgenological conference. Peter Bent Brigham Hospital.

WEDNESDAY, MARCH 1

*12:00 m. Clinicopathologica conference. Children's Hospital.

*Open to the medical profession.

MARCH 2. New England Hospital for Women and Children. Page 208.

MARCH 8. New England Pediatric Society. Page 184, issue of February 10.

MARCH 9. Slipping Rib Cartilage. Diagnosis and Treatment. Dr. John F. Holmes. Pentucket Association of Physicians. 8:30 p.m. Haverhill.

MAY 23 and 24. Massachusetts Medical Society. Annual meeting, Hotel Statler, Boston.

JUNE 12–16. American Medical Association. Annual meeting, Chicago. Page 798, issue of November 18.

DISTRICT MEDICAL SOCIETIES

SUFFOLK

MAY 4. Censors' Meeting. Page xiii, issue of February 10.

WORCESTER

MARCH 8. St. Vincent Hospital, Worcester.

APRIL 12. Hahnemann Hospital, Worcester.

MAY 10. Annual meeting.

BOOKS RECEIVED

The receipt of the following books is acknowledged, and this listing must be regarded as a sufficient return for the courtesy of the sender. Books that appear to be of particular interest will be reviewed as space permits. Additional information in regard to all listed books will be gladly furnished on request.

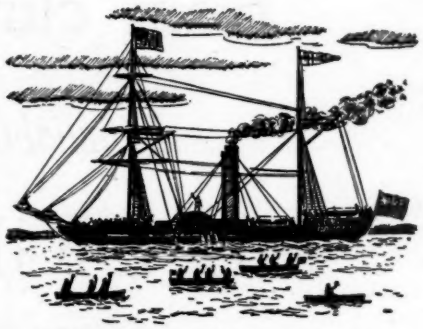
The Condensed Chemical Dictionary. Compiled and edited by the editorial staff of the Chemical Engineering Catalog, Francis M. Turner, editorial director. Third edition, completely revised and enlarged under the supervision of Thomas C. Gregory, editor. 8°, cloth, 756 pp. New York: Reinhold Publishing Corporation, 1942. $12.00.

This edition of a standard reference work has been completely revised and enlarged, particularly for wartime needs. It contains a mass of information on subjects commonly asked for at the present time, among which may be mentioned plastics, organic solvents, poison gases, vitamins and new drugs. Complete descriptions are given of thousands of chemicals, raw materials and manufacturing processes. Over 18,000 items are listed. The book is recommended for reference purposes and should be on the shelves of all those having to do with drugs and chemicals.

Ten Lessons on Meat for Use in Schools. Sixth edition. 8°, paper, 136 pp., with 44 illustrations. Chicago: Department of Home Economics, National Live Stock and Meat Board, 1943. 10 cents.

This large pamphlet, especially designed for teaching in the schools and now in the sixth edition, can be used to advantage in the home. It deals with the structure, identification and selection and with the methods of cooking various meats. There is a chapter on the so-called "variety meats" and another on the art of carving.

The Ship is different today...



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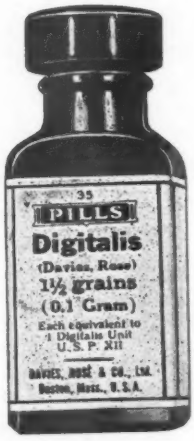

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These freshly prepared, standardized pills are put up in bottles of 35, forming a convenient package for the physician's prescription, obviating the necessity of rehandling.

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